Correspondence

Letters to the editor should be signed by all authors, typewritten in double spacing, and not exceed 600 words of text excluding references. References should be in the Vancouver style. Over-long letters may be shortened without reference to the authors unless it is specifically stated that they may not.

Passive smoking and passive thinking

Mr Lee's objection to the evidence on passive smoking (NZ Med J 1989; 102: 539) hinges on his theory that misclassification of a proportion of smokers as nonsmokers might explain the observed association between passive smoking and lung cancer. However his own book on the subject [1], which he cited, is itself a 100-page monument to bias. In it, he makes an exhaustive exploration of the possibility that smokers are misclassified as nonsmokers, while completely ignoring the fact that the smoking habits of the spouse are equally likely to be misclassified, thus biasing the relative risk estimate towards 1.0. Given the widespread exposure to passive smoking in society, it is likely that epidemiologic studies so far have underestimated the magnitude of risk.

Judging by his remarks on Hirayama's study [2], Mr Lee does not appear to have realised that the age and occupation-standardised rate ratios for ischaemic heart disease have been reported for 17 year follow up [2]. The age and occupation-standardised figures were similar to the rate ratios standardised for age only [2]. We have a complimentary copy of this paper available should Mr Lee wish to read it.

As with Mr Lee, the views we express are always our own. However, we do not receive any fees when we express them. Perhaps this helps clarify the issue of misclassification, which is really very straightforward, and almost invariably leads to an underestimation of the passive smoking effect.

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1 Lee PN. Misclassification of smoking habits and passive smoking. A review of the evidence. International Archives of Occupational and Health. Supplement. Heidelberg: Springer Verlag, 1988.
2 Hirayama T. Lung cancer in Japan: effects of nutrition and passive smoking. In: Mizell M, Correa P, eds. Lung cancer: causes and prevention. New York: Verlag Chemie International, 1984: 175-95.

Treatment of hypertension

Kawachi and Purdie's reply (NZ Med J 1989; 102: 540) to my letter [1] raises important issues in the debate about the benefits of treating hypertension. Two main points require serious consideration. The first is contained in the advice that I should consult their data pertaining to treatment at a blood pressure greater than 100 mmHg [2], and to accept that these data represent the expected gain with treatment if hypertension is treated according to the recently published guidelines. This is wrong. The advice fails to recognise that the definition of hypertension by trial protocols is quite different from that suggested by recent guidelines, and it is not difficult to show that trial patients are unrepresentative of the larger population and are at lower risk even at equal levels of blood pressure because of exclusion criteria and the method of selection. A patient whose diastolic blood pressure is 100 mmHg after several readings taken over a reasonable time interval is at higher risk than if selected from a low risk population on the basis of screening measurements. In the placebo treated group of the MRC trial, whose records I am currently studying, the unadjusted cardiovascular event rate at entry for those with diastolic blood pressure greater than 100 mmHg (n=3022) was 8.1/1000/yr, but in those with the same blood pressure 3 months into the trial (n=1198) (equivalent to a definition of hypertension more in keeping with the guidelines) the corresponding figure was 10.3 (Millar and Lever, unpublished). This illustrates that current guidelines have the effect of identifying a subset of patients with a greater risk, thereby optimising the efficiency of treatment measured as the number of patients treated per event avoided, in this case 373 versus 116 respectively. These figures are much lower than those presented by Kawachi and Purdie [2].

The second point relates to the long term benefits of treatment. This is a complex and important issue which cannot be fully addressed here. Suffice to say that Kawachi and Purdie's suggestion that treatment can be deferred until the blood pressure rises to unacceptable levels not only implies an arguable value judgment on their part but is illogical and not supported by any evidence that I know, though it has to be acknowledged as a possibility. No trial has shown that deferred treatment confers benefit (none has been designed to do so), and although left ventricular hypertrophy can regress with some forms of treatment (generally the expensive ones!) there is evidence that permanent ultrastructural changes occur in the myocardium. Common sense suggests that treatment should be started as soon as a proper diagnosis according to current guidelines is made.

I noted the subtle change in the identity of the decision maker from the doctor to the patient in the case of my hypothetical 30 year old. This raises interesting questions. Whose is the responsibility if the patient makes the wrong decision? Is it realistic to expect him to review his decision at regular intervals, and if so, on what grounds will he reverse it? Will this happen before or after the onset of hemiplegia or dyspnoea?

The drug side effects are important for the patient and as a determinant of the overall ratio of costs to benefits. I believe the point I was making is clear enough and I leave it to practitioners to decide from their experience whether side effects such as impotence are reversible on withdrawal of the offending agent or not.

A previous paper from my correspondents' department has compared (unfavourably) the cost of treating hypertension with cardiac transplantation [3], and the clear implication from their publications is that they regard the treatment of mild hypertension as prohibitively expensive. We have agreed with their conclusion, up to a point [4,5], but have provided cost benefit analyses based on both trials of treatment and current management guidelines. It would be instructive to see similar calculations from Kawachi and Purdie.

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1 Millar JA. Treatment of hypertension. NZ Med J 1989; 102: 478.
2 Kawachi I, Purdie G. The benefits and risks of treating mild to moderate hypertension. NZ Med J 1989; 102: 577-9.
3 Malcolm L, Jackson R, Kawachi I, Bonita R. Is the pharmacological treatment of mild to moderate hypertension cost effective in stroke prevention? NZ Med J 1988; 101: 167-71.
4 Millar JA, Hansen PC. The economics of treating mild hypertension (Letter). NZ Med J 1988; 101: 275.
5 Millar JA, Hansen PC. Economic costs and benefits of treating mild hypertension: results from a cross sectional model. NZ Med J 1988; 101: 623-5.

Diet and behaviour

I write in response to the leading article, Diet and Behaviour (NZ Med J 1989; 102: 499). I am the mother of three children as well as being a general practitioner. Our 3½ year old daughter is food sensitive, and I have no doubt that the ingestion of foods or additives that disagree with her cause deteriorating behaviour, dark circles under her eyes, night waking (1.30 am-4.30 am), loss of appetite, increased thirst, vulvitis and joint pains. These reactions have been confirmed on several occasions by (often inadvertent) challenge tests. I have been manipulating her diet for a year with excellent results and improved sleep, and contest that, far from a negative effect, it has developed great responsibility and concern in her 5 year old sister that she should not be exposed to foods that make her ill.

Critical observation has long been the backbone of medical practice. Accurate deductions have been made before the process in question was understood. We need look no further than the development of vaccination by Edward Jenner, the correlation between handwashing and puerperal infection noted by Ignaz Semmelweiss and the discovery of penicillin by Alexander Fleming.

Parents and teachers are the people best able to assess the behaviour of children. Early and subtle behaviour changes are unlikely to be noticed by independent researchers who do not know the children. We also are not aware of the cumulative effect of these small behavioural changes on the educational life of the